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Amicarbazone, a New Photosystem II Inhibitor

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Amicarbazone is a new triazolinone herbicide with a broad spectrum of weed control. The phenotypic responses of sensitive plants exposed to amicarbazone include chlorosis, stunted growth, tissue necrosis, and death. Its efficacy as both a foliar- and root-applied herbicide suggests that absorption and translocation of this compound is very rapid. This new herbicide is a potent inhibitor of photosynthetic electron transport, inducing chlorophyll fluorescence and interrupting oxygen evolution ostensibly via binding to the Q_B domain of photosystem II (PSII) in a manner similar to the triazines and the triazinones classes of herbicides. As a result, its efficacy is susceptible to the most common form of resistance to PSII inhibitors. Nonetheless, amicarbazone has a good selectivity profile and is a more potent herbicide than atrazine, which enables its use at lower rates than those of traditional photosynthetic inhibitors.

Nomenclature: Amicarbazone; photosystem inhibitor, CAS No. 129909-90-6, BAY MKH 3586; atrazine, CAS No. 1912-24-9.

Key words: Herbicide mode of action, photosynthesis, photosystem II, herbicide resistance.

Amicarbazone, 4-amino-*N*-(1,1-dimethylethyl)-4,5-dihydro-3-(1-methylethyl)-5-oxo-1*H*-1,2,4-triazole-1-carboxamide, is a triazolinone herbicide with a broad spectrum of weed control in corn (*Zea mays* L.) and sugar cane (*Saccharum officinarum* L.) fields (Philbrook et al. 1999). The primary weeds targeted by amicarbazone include velvetleaf (*Abutilon theophrasti* Medik.), common lambsquarters (*Chenopodium album* L.), pigweed species (*Amaranthus* spp.), common cocklebur (*Xanthium strumarium* L.), and morningglory species (*Ipmoea* spp.). In sugar cane, amicarbazone also controls painted wild poinsettia (*Euphorbia heterophylla* L.), morningglories, southern sandbur (*Cenchrus echinatus* L.) and marmaladegrass [*Brachiaria plantaginea* (Link) Hitch.] (Senseman 2007).

Amicarbazone provides residual weed control in reduced- and zero-tillage corn production systems and can be applied in corn as a preplant or PRE. It is also registered for PRE or POST application in sugar cane fields. Amicarbazone can also be applied in combination with other herbicides to broaden the spectrum and efficacy of weed control (Philbrook et al. 1999). For example, mixtures of trifloxysulfuron, ametryn, and amicarbazone provided broader weed control without injuring sugar cane and ratoon cane with residual activity over 4 mo after treatment (Seeruttun et al. 2008).

Due to the large amount of straw residues typically present in sugar cane production, the interception of amicarbazone by straw has been tested. Overall, 5 tons of straw per hectare or more trapped nearly all of the applied herbicide, and increases in the time interval between herbicide application and first rain decreased leaching (Cavenaghi et al. 2007). Furthermore, the effect of straw on the efficacy of amicarbazone against marmaladegrass, signalgrass (*Brachiaria decumbens* Stapf), viola (*Ipomoea grandifolia* L.), and purple nutsedge (*Cyperus rotundus* L.) was tested. The highest control indices were obtained when amicarbazone was applied on the straw followed by simulated rainfall or when applied directly in the soil (Negrisoli et al. 2007).

Amicarbazone is listed as an inhibitor of photosynthesis (Senseman 2007) and is described to cause chlorosis, stunted growth, tissue necrosis, and death of susceptible plant species (Philbrook et al. 1999). However, no data on its effect on photosynthesis or photosystem II (PSII) are available. Herein, we compare the in vivo herbicidal activity of amicarbazone and atrazine on selected broadleaf and grass weeds and corn and their relative effect on in vivo photosynthetic efficiency. The relative potency of amicarbazone and atrazine on photosystem II–driven photosynthetic oxygen evolution from isolated thylakoid membranes of corn, velvetleaf, and wild-type and triazine-resistant redroot pigweed (*Amaranthus retroflexus* L.) was investigated.

Materials and Methods

Growth of Plants. Seeds of corn hybrid DKC 61-22, velvetleaf, large crabgrass [*Digitaria sanguinalis* (L.) Scop.], barnyardgrass [*Echinochloa crus-galli* (L.) Beauv], and wild-type and triazine-resistant redroot pigweed were purchased from commercial dealers.[1]

Seeds were planted in 15-cm plastic round pots containing Metro-Mix 350 potting soil[2] and the seedlings were thinned to about 10 plants per pot after germination. Pots were maintained in the greenhouse without supplemental lighting for the duration of the experiments and hand watered from the bottom as needed. Average daytime and nighttime temperatures were 28 and 19 C, respectively.

Assessing the Effect of Amicarbazone and Atrazine on Plant Biomass. Plants were allowed to grow for 4 wk before being treated with either amicarbazone or atrazine with a Generation III Research track sprayer[3] equipped with a XR80015 Teejet nozzle[4] at a delivery rate of 375 L ha^{-1}. Amicarbazone was applied at 62.5, 125, 250, 500, and 1,000 g ha^{-1}, and atrazine was applied at 125, 250, 500, 1,000, and 2,000 g ha^{-1}. All treatments were applied with 0.2% crop oil concentrate,[5] and the controls received water with the adjuvant only. At the end of the experiments, the areal portions of the plants were harvested, placed in paper bags, and dried at 80 C for 48 h. All treatments were repeated four times and the experiment was organized in a split-plot design, by herbicide concentration. The data consist of the dry weight (DW) at the end of the experiments.

Assessing the Effect of Amicarbazone and Atrazine on Photosynthetic Efficiency. Photosynthetic quantum yield

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(Y) and electron transport rate (ETR) were measured with the use of a pulse-modulated fluorometer.[6] The instrument was set on kinetic mode and adjusted so that the initial Ft (instantaneous fluorescence signal) value in the control samples was approximately 210. Quantum yield was determined by the following light treatment: Each cycle consisted of a 0.8-s pulse of saturating light generated with a laser diode actinic source to saturate PSII, followed by a 1-s far-red light pulse used to reoxidize PSII, and a 20-s delay to allow PSII to regain steady-state conditions. A total of eight cycles were performed for each sample. ETR values were expressed as percents of the ETR average values observed in control treatments.

A time-course experiment was performed by measuring induced fluorescence on intact leaves at predetermined time intervals (up to 24 h) following foliar application of 500 g ha^{-1} amicarbazone or 1,000 g ha^{-1} atrazine. Both treatments were applied with 0.2% crop oil concentrate,[4] and the controls received water with the adjuvant only. The experiment was carried out with four replications.

In a second experiment, the root systems of corn, velvetleaf, and large crabgrass plants were submerged into solutions containing amicarbazone (10, 33, 100, and 300 μM) or atrazine (44, 133, 400, and 1,200 μM). The experiment was carried out with four replications. Each plot corresponded to one disposable plastic tube filled with 30 ml of water or a solution containing amicarbazone or atrazine and one plant. All tubes were weighed at 0 (prior to the treatment), 3, 6, and 24 h after treatment at the time when ETR measurements were made in the middle portion of two leaves per plant. The water losses were corrected by subtracting the water losses in tubes without plants. At the end of the experiment the leaf area of each plant was measured as described by Kinoshita et al. (2008). The total transpiration was estimated by the reduction in the weight of each plot at each interval. The amounts of the herbicides intercepted by the roots were calculated by multiplying the accumulated water consumptions and the herbicide concentrations. By dividing these values by the leaf area of each plant, it was possible to estimate the accumulated herbicide intercepted (AHI, expressed in nmol cm^{-2}) by the roots at each interval, as a function of the leaf area. The data were organized in an increasing sequence of AHI values for each herbicide.

Extraction of Photosynthetic Thylakoid Membranes. Thylakoid membranes were isolated from corn, velvetleaf, and wild-type, and triazine-resistant pigweed as described by Rimando et al. (1998), except that the thylakoid membranes were further purified by sucrose gradient centrifugation (Dayan et al. 1998). Thylakoid membranes were diluted to 4 mg of chlorophyll ml^{-1} for the oxygen evolution experiments.

Oxygen Evolution Assays. O_2 evolution was initiated under saturating light conditions (10 mmol m^{-2} s^{-1} PAR) with the use of a fiber-optic light source and measured polarographically with the use of a computer-controlled oxygen probe.[7] The reaction assay buffer consisted of 800 mM sucrose, 50 mM Mes-NaOH buffer (pH 6.2), 15 mM $CaCl_2$, and 1 mM K_3Fe-$(CN)_6$. All assays were performed at 30 C. Amicarbazone and atrazine were diluted in acetone, and control treatments received the same concentration of solvent (less than 1% v/v). Membrane preparations were incubated with test compounds (0 to 3 μM) on ice for 20 min prior to the assay. The assay was initiated by addition of thylakoid membranes to the reaction assay buffer, and the rate of oxygen evolution was measured for 20 s over the linear portion of the curve. Data are expressed as relative activity.

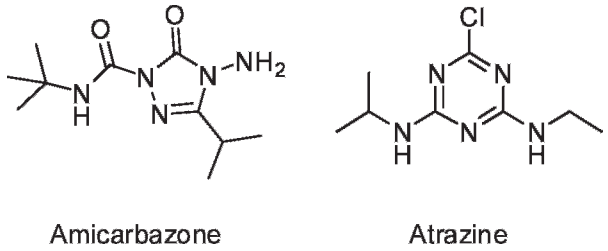

Figure 1. Structures of amicarbazone and atrazine.

Statistical Analysis. Data from dose–response experiments were analyzed with the add-on package for dose–response curves (Ritz and Streibig 2005) for R version 2.9.0 (R-Development-Core-Team 2009) with the use of the following logistic function. The dose–response and time-course experiments were modeled according to Equation 1.

$$y = c + \frac{d - c}{1 + \left(x / V_{50}\right)^{b}} \quad [1]$$

where y represents either biomass accumulation (DW), photosynthetic electron transport (ETR), or photosynthetic oxygen evolution (O_2), x represents dose of herbicide or time in hours, d denotes the upper and c the lower limits, and V_{50} denotes the dose (either EC_{50} or I_{50}) or the time required to halve the response y between the d and c.

The dose–response curves of the effect of the soil-applied herbicides on ETR of the test species were fitted to Equation 2.

$$y = \frac{1}{ax + b} \quad [2]$$

where y represents ETR values, a and b determine the way in which ETR decreases with herbicide interception by the roots, and x represents AHI values.

Results and Discussion

Many compounds based on the triazolinone backbone have been developed as herbicides that target protoporphyrinogen oxidase, a key enzyme in heme and chlorophyll synthesis (Dayan et al. 1996, 1997a,b, 1998; Luo et al. 2008; Theodoridis et al. 1992). Propoxycarbazone–sodium, a sulfonyltriazolinone analog of amicarbazone, was recently introduced for the management grass weeds in cereals (Mueller 2002). It inhibits branched-chain amino acids by targeting the enzyme acetolactate synthase (Senseman 2007). Amicarbazone (Figure 1), on the other hand, is reportedly an inhibitor of photosynthetic electron transport but no data have been reported to document this mechanism of action. However, amicarbazone possesses a sp^2 hybrid C–NH, which is a well-known structural feature shared by most PSII inhibitors (Trebst 2007).

The herbicidal effect of amicarbazone was compared to that of atrazine on corn and selected weed species. Amicarbazone was more potent on the broadleaf weeds than the mono-

Table 1. Inhibition of growth by amicarbazone and atrazine on corn, velvetleaf, wild-type pigweed, crabgrass, and barnyardgrass.

Compound	Corn	Velvetleaf	Pigweed	Crabgrass	Barnyardgrass
	EC_{50} (g ha^{-1})[a]				
Amicarbazone	458 ± 200	42.9 ± 1.7	42.8 ± 4.1	238.5 ± 67.9	166.3 ± 23.4
Atrazine	2,570 ± 208	459 ± 80.6	45.1 ± 4.9	1,027 ± 380	407.8 ± 39.0

[a] EC_{50} = concentration of herbicide for 50% inhibition of dry weight accumulation. Data were obtained with the use of Equation 1 (see Materials and Methods section).

cytoledonous weeds. The EC_{50} of amicarbazone on velvetleaf and pigweed was 10-fold lower than on corn, indicating a good safety margin (Table 1). In contrast, the grass weeds crabgrass and barnyardgrass required higher rates of amicarbazone, EC_{50} values at 238.5 and 166.3 g ha^{-1}, respectively. The performance of amicarbazone was superior to atrazine on all the weeds tested, but especially on velvetleaf and crabgrass (Table 1). However, corn was more sensitive to amicarbazone than to atrazine (Table 1).

Because amicarbazone is reported to be a PSII inhibitor, its effect on photosynthetic electron transport (ETR) was measured by monitoring the induced fluorescence resulting from either foliar or root applications. Time-course studies clearly demonstrated that the effect of foliar-applied amicarbazone on ETR was much more rapid on velvetleaf and crabgrass than on corn (Figure 2A). The weeds' ETRs were nearly completely inhibited within 8 h of application, whereas corn retained more than 30% of its photosynthesis after 24 h. Corn was even more tolerant to atrazine, with more than 70% of its photosynthetic ETR remaining 24 h after application (Figure 2B). Photosynthesis of crabgrass was very sensitive to atrazine, whereas that of velvetleaf was more tolerant. Consistent with the biomass data (Table 1), amicarbazone had a stronger effect on velvetleaf that atrazine (compare Figures 2A and 2B). This may be due to the fact that velvetleaf possesses a glutathione-*S*-transferase that has the capacity to metabolize atrazine rapidly (Anderson and Gronwald 1991).

Foliar absorption of amicarbazone was not quantified with the use of conventional radiolabeled techniques, but our data nonetheless provides some evidence that amicarbazone is readily absorbed through the foliage and subsequently has a strong inhibitory effect on photosynthesis. The difference in response between the various species tested may be due to different rates of absorption and/or metabolism, but that remains to be determined.

Amicarbazone is also commercialized as a PRE herbicide. Application of this inhibitor to the roots of the plants species tested resulted in inhibition of ETR in the foliage (Figure 3A), which indicates that this herbicide is readily taken up by roots and translocated to the foliage. This dose–response study shows that the ETR of both crabgrass and velvetleaf were more affected than that of corn. A similar study carried out with atrazine suggests that its effect is somewhat lower than that of amicarbazone (Figure 3B). Consistent with the biomass data from the greenhouse study (Table 1), the ETR of corn was much less affected by atrazine than by amicarbazone (Figure 3B). This may be due to the well-established ability of corn to detoxify atrazine rapidly via glutathione-*S*-transferase conjugation (Shimabukuro et al. 1971). Metabolic degradation of amicarbazone in corn (or any other plant species) is not yet known.

In light of the strong inhibition of photosynthetic ETR by amicarbazone, we tested whether amicarbazone also inhibited photosynthetic oxygen evolution. Amicarbazone was indeed a potent inhibitor of PSII in isolated thylakoid membranes of corn and velvetleaf. The effect of amicarbazone on oxygen evolution was approximately 10 times stronger than atrazine on velvetleaf, whereas it was approximately 3 times weaker than atrazine on corn oxygen evolution (Table 2). This relative potency of amicarbazone and atrazine on corn oxygen evolution is interesting because it is the reverse of the effect on corn growth and in vivo leaf ETR. This may be an indication that amicarbazone is absorbed by the treated leaves faster than atrazine and/or that its metabolic degradation is slower.

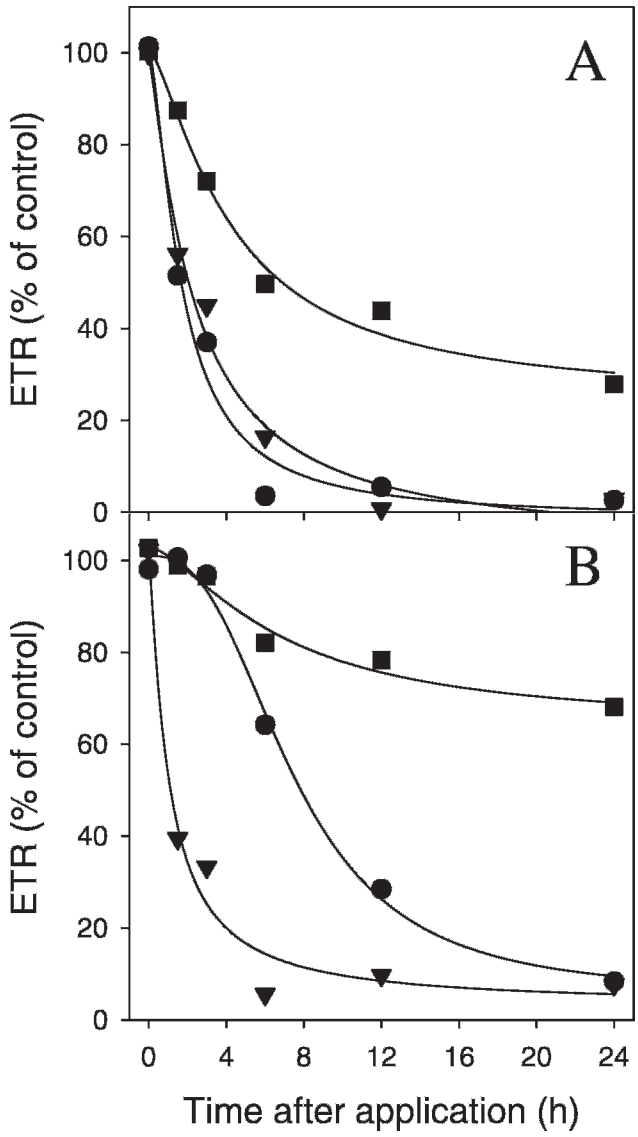

Figure 2. Effect of foliar-applied (A) amicarbazone (500 g ha^{-1}) and (B) atrazine (1,000 g ha^{-1}) on photosynthetic electron transfer rate (ETR) of corn (■), and the monocotyledonous weed crabgrass (▼), and the dicotyledonous weed velvetleaf (●). The data were fitted with the use of Equation 1 (see Materials and Methods section).

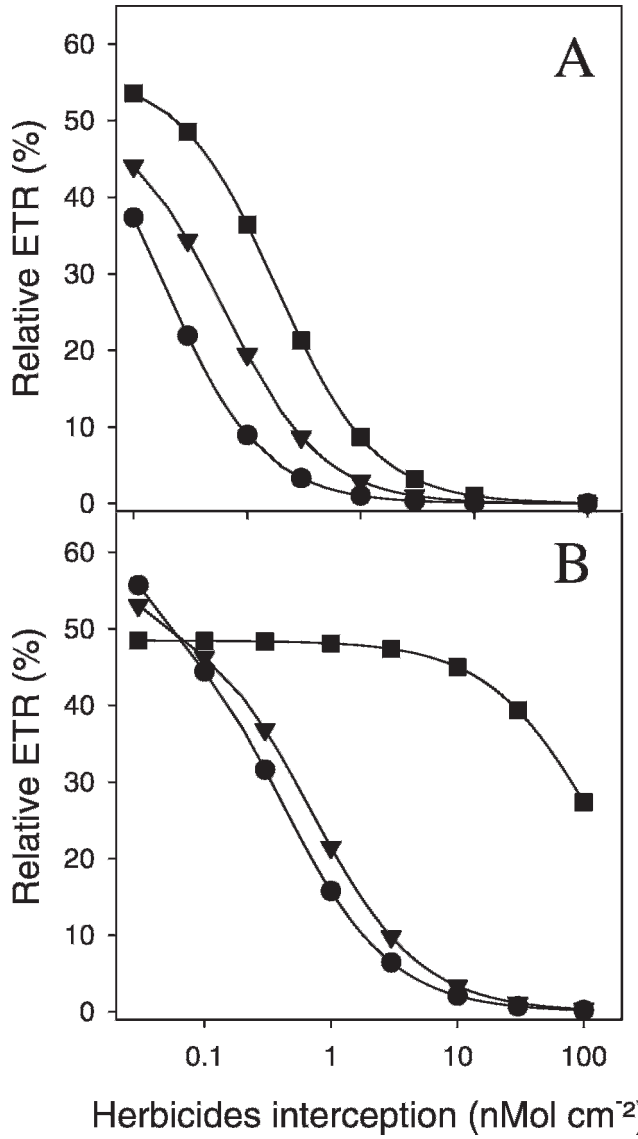

Figure 3. Correlation between root-applied (A) amicarbazone and (B) atrazine photosynthetic electron transfer rate (ETR) of corn (■), and the monocotyledonous weed crabgrass (▼), and the dicotyledonous weed velvetleaf (●). The data were fitted with the use of Equation 2 (see Materials and Methods section).

The strong inhibition of oxygen evolution and rapid induction of chlorophyll fluorescence confirms that amicarbazone interacts with the Q_B binding site of plastoquinone on PSII (Hess 2000), which is the same molecular target site as a number of herbicide classes such as the triazines, uracils, benzonitriles, substituted ureas, phenylcarbamates, triazinones, and pyridazinones (Senseman 2007). Therefore, a final issue was to determine whether the efficacy of this new triazolinone herbicide was susceptible to the most common form of resistance to PSII inhibitors. Indeed, resistance to inhibitors of PSII first appeared nearly 50 yr ago, when common groundsel (*Senecio vulgaris* L.) resistance to simazine was confirmed (Ryan 1970) and it has now been reported in at least 68 weed species (Heap 2009). Most *s*-triazine resistant biotypes show a high level of cross-resistance to other *s*-triazine herbicides, a lower level of resistance to the triazinones (e.g., metribuzin), but no cross-resistance to phenylurea herbicides (e.g., linuron)(Oettmeier 1999).

Although all of these compounds inhibit PET by interacting with the Q_B binding site, it has been shown that they interact with different amino acids within the binding pocket (Draber et al. 1991; Sobolev and Edelman 1995; Trebst and Draber 1986). The most common mechanism of target-site resistance to PSII inhibitors involves a Ser_{264} to Gly mutation in the D1 protein. The binding affinity of triazines to this mutated site is greatly reduced, but it has no effect on the affinity of substituted urea herbicides and other PSII electron transport inhibitors (e.g., quinone-type inhibitors) (Dayan et al. 2009; Draber et al. 1995).

Table 2. Inhibition of photosynthetic oxygen evolution by amicarbazone and atrazine on isolated thylakoid membranes of corn, velvetleaf, and WT and R pigweed.[a]

Compound	Corn	Velvetleaf	WT pigweed	R pigweed
	I_{50} (μM)[b]			
Amicarbazone	0.66 ± 0.11	0.06 ± 0.01	0.16 ± 0.02	≫ 20
Atrazine	0.23 ± 0.03	0.78 ± 0.21	0.62 ± 0.11	≈ 20

[a] Abbreviations: WT, wild-type; R, triazine-resistant.
[b] I_{50} = concentration of inhibitor for 50% inhibition of the rate of oxygen evolution produced by isolated thylakoid membranes. Data were obtained with the use of Equation 1 (see Materials and Methods section).

Amicarbazone and atrazine were tested on thylakoid membranes isolated from wild-type and triazine-resistant (Ser_{264} mutant) pigweed. As reported many times, the potency of atrazine was at least 32 times lower on the photosynthetic oxygen evolution from the resistant pigweed than the wild-type (Table 2 and Figure 4). Consistent with the effect on velvetleaf, amicarbazone was nearly four times more active than atrazine on the wild-type pigweed thylakoids. On the other hand, the level of resistance was stronger than that observed with atrazine. Therefore, biotypes containing the Ser_{264} mutation are highly resistant to amicarbazone (and other triazolinone)(Table 2 and Figure 4).

This is consistent with other work investigating the effect of various PSII mutations on the binding of triazinone-type PSII herbicides (e.g., metamitron) to the D1 binding pocket (Draber et al. 1991). The triazinone compounds were

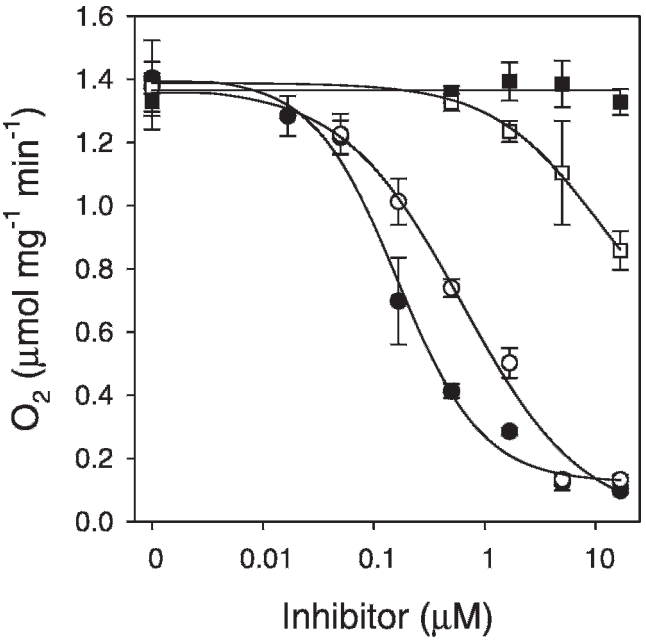

Figure 4. Effect of amicarbazone (black) and atrazine (white) on photosynthetic oxygen evolution of wild-type (circle) and triazine-resistant (square) pigweed. The data were fitted according to Equation 1 (see Materials and Methods section).

stabilized within the binding pocket by two key hydrogen bonding interactions, the free amino group of metamitron participated in a hydrogen bond interaction with Ser_{264}, and the carbonyl group of the triazinone ring was stabilized via a hydrogen bond to the peptide group Phe_{265}. The potency of all triazinones was greatly reduced by the Ser_{264} mutation, which was accounted by the loss of a hydrogen bond between the hydroxyl group of Ser_{264} and the free amino group of the triazinones (Draber et al. 1991). The decrease in the efficacy of amicarbazone on the triazine-resistant pigweed appears to be based on a similar type of interaction, because this triazolinone is structurally related to metamitron in that it also possesses a free amino group and a carbonyl moiety (Figure 1).

In conclusion, amicarbazone is a new selective herbicide for corn and sugar cane production systems. It is more potent than atrazine and therefore can be used at lower rates (Philbrook et al. 1999). Its efficacy as both a foliar and root-applied herbicide suggests that absorption and translocation of this compound is very rapid. Amicarbazone is a potent inhibitor of photosynthetic electron transport. It appears to interact with PSII in a similar manner as the triazines and the triazinones classes of herbicides. As a result, its efficacy is susceptible to the most common form of resistance to PSII inhibitors, which may limit its usefulness in combating ongoing problems with weeds expressing the Ser_{264} mutation in their D1 protein.

Sources of Materials

[1] Seed Service, Leland, MS 38756, USA; DeKalb seed, Monsanto Company, St. Louis, MO 63167; Herbiseed, Twyford, United Kingdom.

[2] Potting soil, Sun Gro Horticulture, Bellevue, WA 98008.

[3] Track sprayer, DeVries Manufacturing, Hollandale, MN 56045.

[4] Teejet nozzle, Spraying Systems Co., Wheaton, IL 60189.

[5] Sanders Low Foam Surfactant, Jimmy Sanders Inc., Cleveland, MS 38732.

[6] Pulse-modulated fluorometer, Model OS5-FL, Opti-Science, Hudson, NH 03051.

[7] DW1 oxygen probe connected to an OMS1 oxygen monitoring system, Hansatech Instrument Ltd., Norfolk, England.

Acknowledgments

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